

Thrombophlebitis and Phlebothrombosis

Venous thrombosis • mechanisms • morphology • clinical consequences

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Learning goals

- Explain how stasis and hypercoagulability produce venous thrombosis.
- Recognize the major anatomic locations and venous thrombus morphology.
- Interpret the often-silent presentation and serious embolic sequelae.
- Apply the pathology to immobilization, malignancy, and inherited thrombophilia.

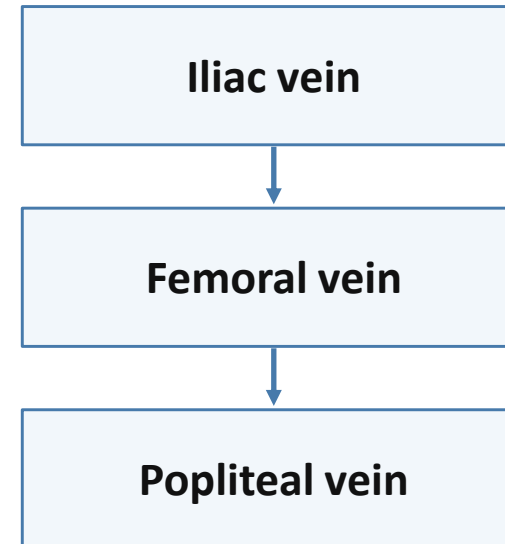
Two terms describe related venous pathology

- Thrombophlebitis and phlebothrombosis are used interchangeably.
- Both refer to venous thrombosis with an associated inflammatory component.
- In clinical reasoning, the location and embolic potential matter more than terminology.

Most venous thromboses involve the deep leg veins

- Deep leg veins account for more than 90% of cases in Chapter 11.
- Proximal deep veins include the popliteal, femoral, and iliac veins.
- These vessels are important sources of pulmonary thromboemboli.

Lower-extremity veins



Deep venous path (schematic)

Simplified named major proximal veins; Not anatomical scale

Superficial and deep venous disease differ clinically

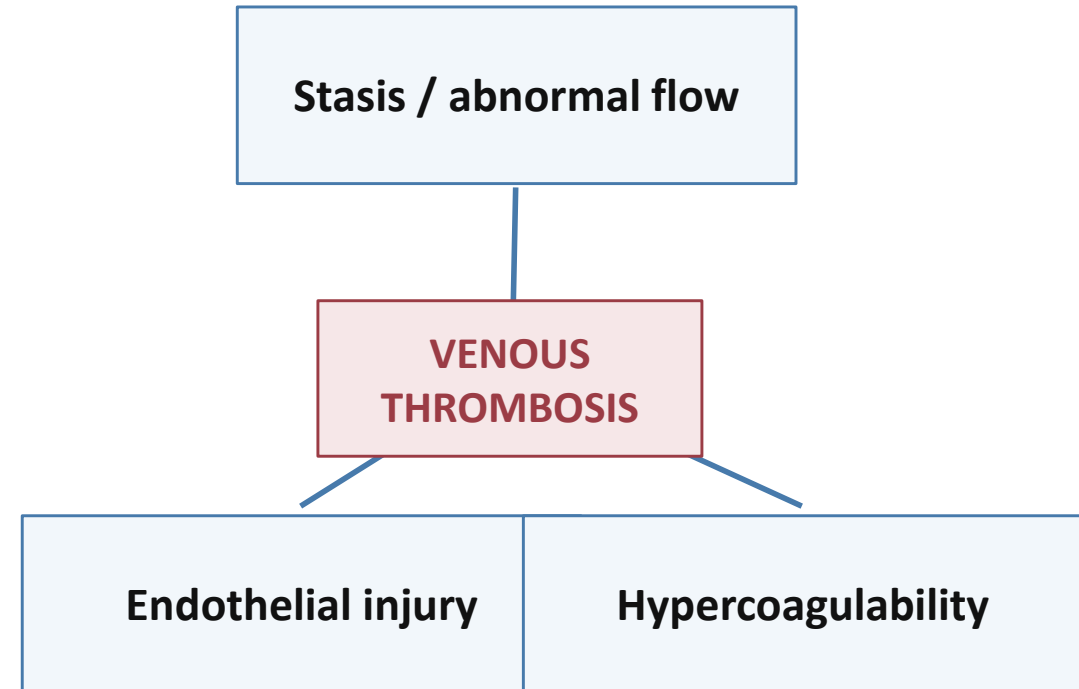
- Superficial saphenous thromboses commonly accompany varicose veins.
- Deep vein thrombosis has a stronger association with pulmonary embolization.
- Both can obstruct venous drainage, but the principal hazard differs.

Feature	Superficial thrombosis	Deep vein thrombosis
Common setting	Varicosities / saphenous veins	Deep leg venous system
Dominant problem	Local tenderness / congestion	Pulmonary embolic potential
Embolization	Uncommon	More frequent

Editable comparison table

Venous thrombosis belongs to the Virchow triad

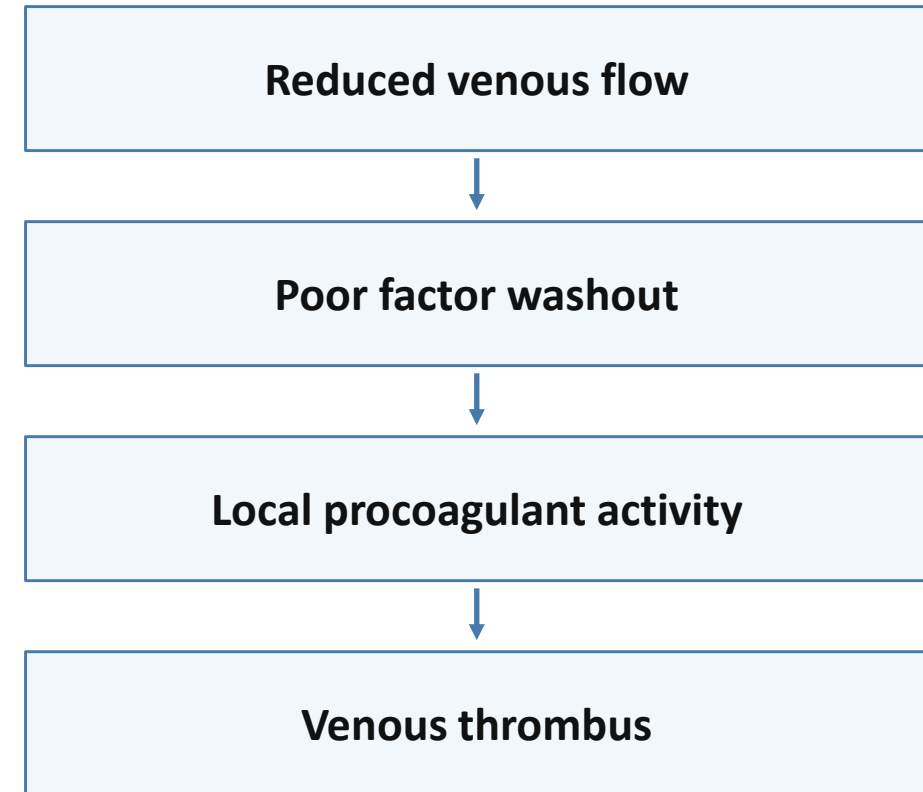
- Endothelial injury or activation
- Abnormal blood flow, particularly stasis
- Hypercoagulability: Inherited or acquired
- More than one component may operate in the same patient.



Virchow triad: Interacting factors, not exclusive causes

Stasis favors thrombosis in low-flow veins

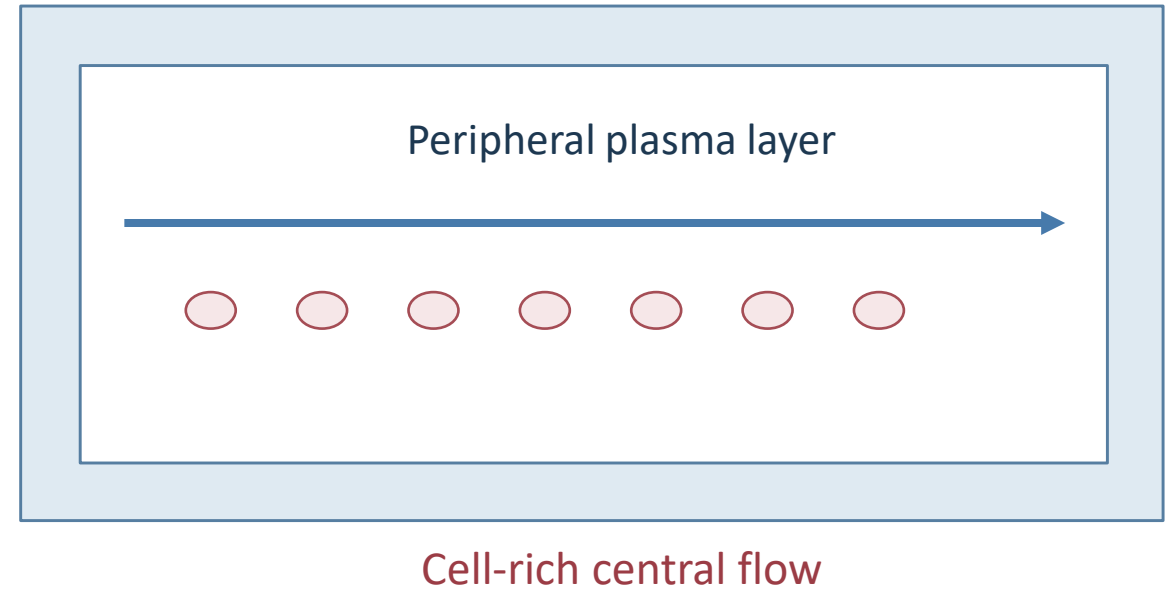
- Sluggish venous blood permits local accumulation of activated coagulation factors.
- Washout by incoming blood becomes less effective.
- Flow disturbances can also promote endothelial activation and leukocyte adhesion.



Original editable causal schematic

Laminar flow normally separates cells from endothelium

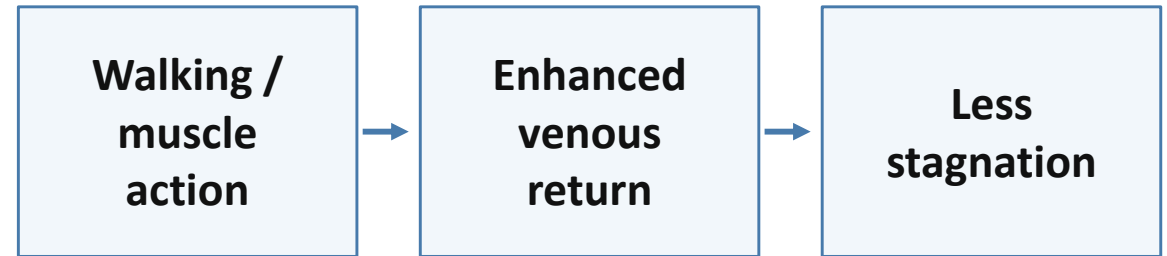
- Circulating cellular elements tend to remain in the central stream.
- A peripheral plasma layer limits sustained cellular contact with endothelium.
- Stasis and disturbed flow weaken the protective effects of normal circulation.



Flow distribution schematic, not microscopy

The skeletal-muscle pump limits leg venous stasis

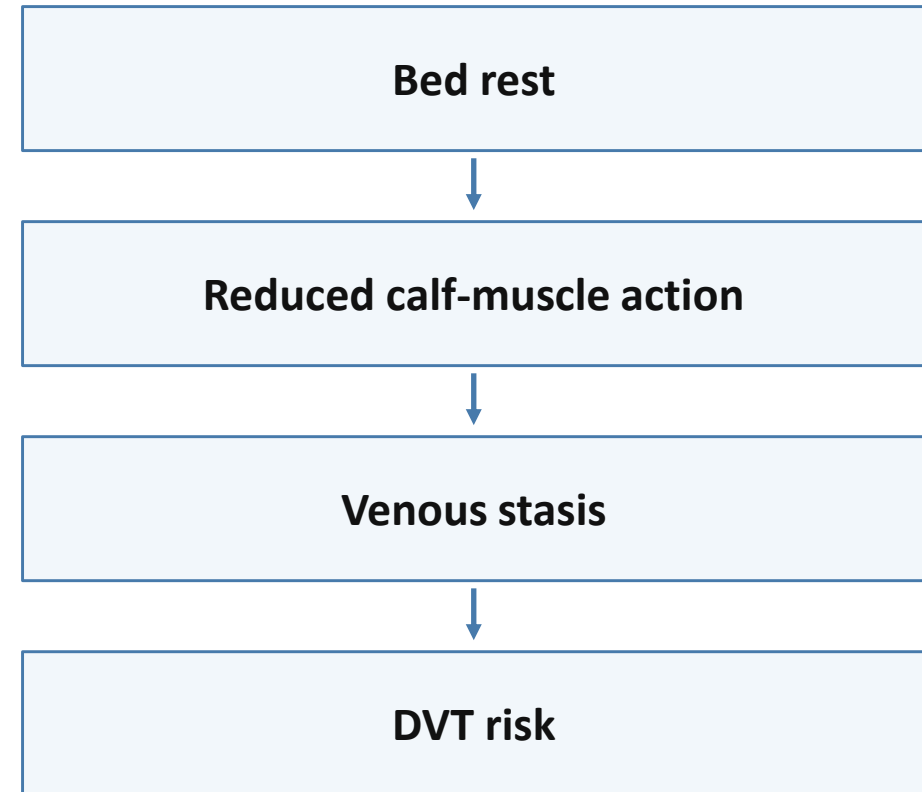
- Leg-muscle contractions assist venous return against gravity.
- Immobilization reduces the muscle-pump contribution.
- The resulting slow venous flow creates a permissive thrombotic environment.



Original editable causal schematic

Prolonged bed rest is a major risk context

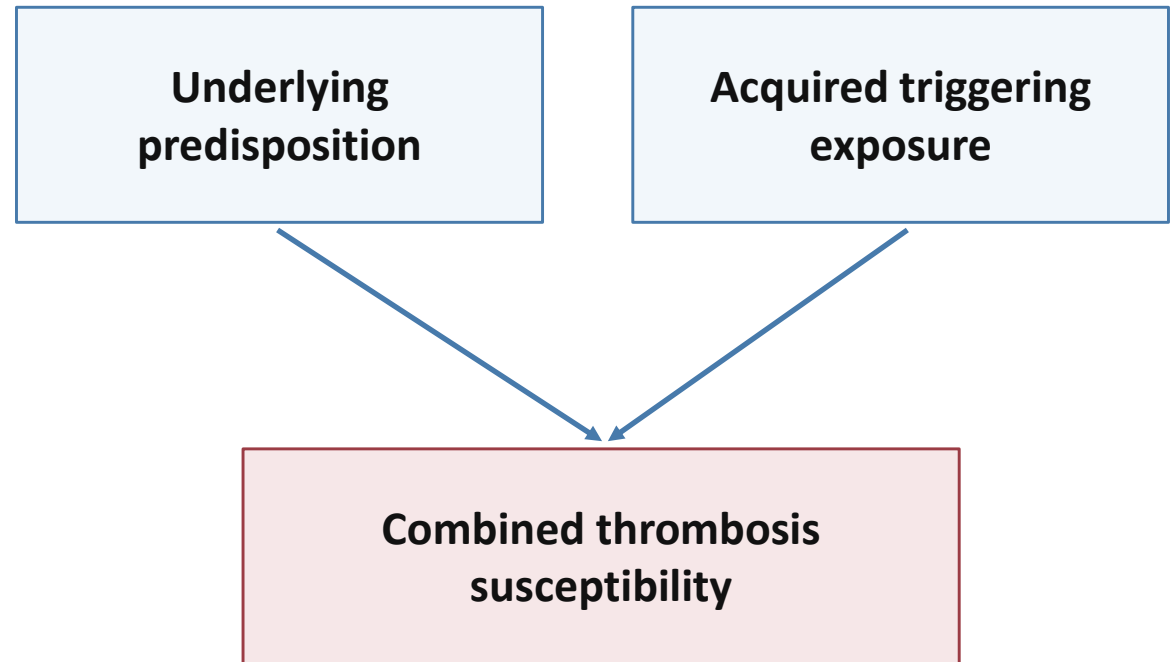
- Immobility is identified as the most common cause of lower-extremity DVT in Chapter 11.
- Reduced leg movement causes venous stasis.
- Bedridden patients may have very few local warning symptoms.



Original editable causal schematic

Long-distance sitting reproduces the stasis mechanism

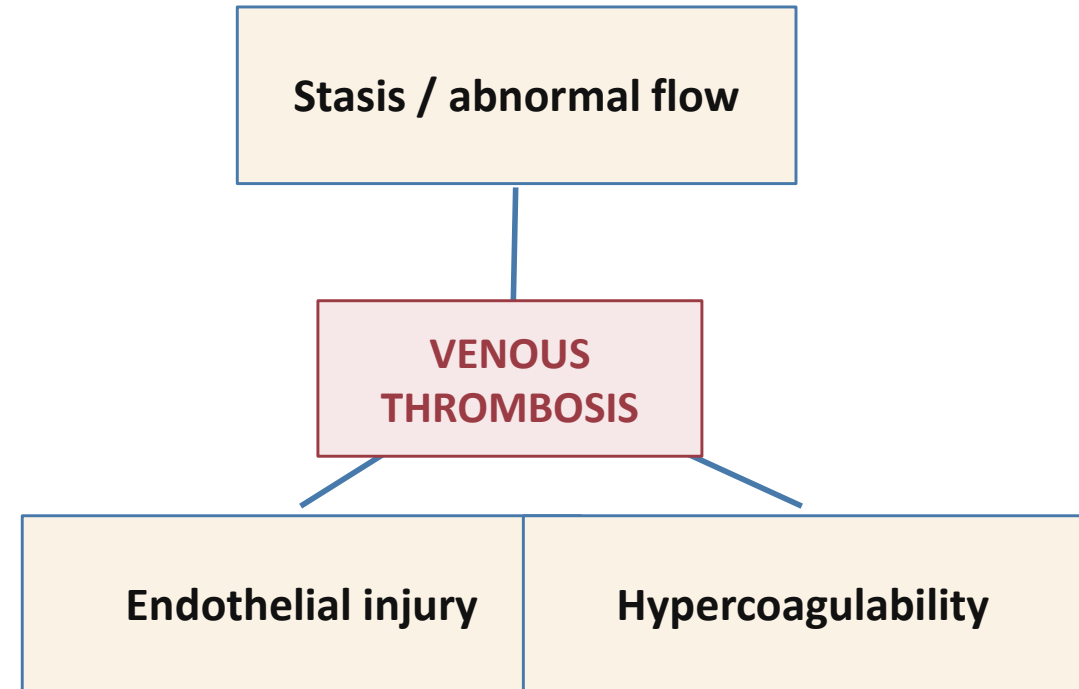
- Long airplane or automobile journeys can involve extended sitting.
- The shared exposure is prolonged reduction in leg movement.
- An inherited predisposition can combine with this acquired risk.



Multi-hit model; Relationships are qualitative

Postoperative DVT has multiple mechanistic drivers

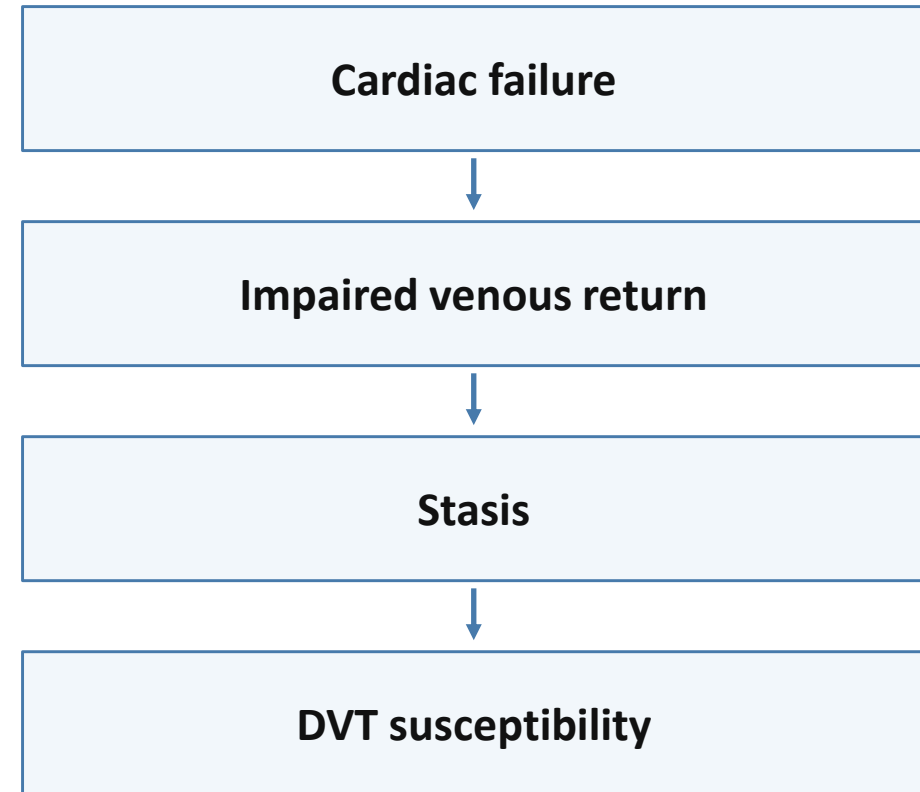
- Postoperative immobility suppresses venous return.
- Tissue injury can release procoagulant activity.
- Vascular injury and alterations in fibrinolysis further shift the balance toward clotting.



Virchow triad: Interacting factors, not exclusive causes

Congestive heart failure promotes venous stasis

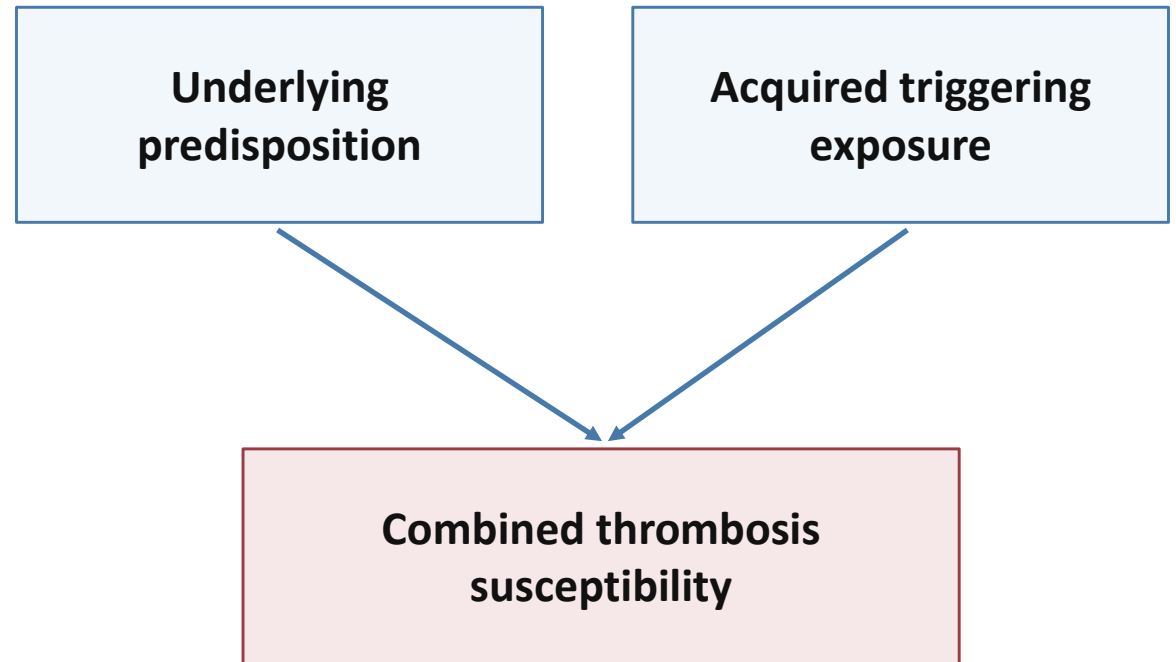
- Reduced effective forward flow impairs venous return.
- Stagnant leg venous blood favors coagulation.
- Heart failure adds thrombotic risk even without a local leg injury.



Original editable causal schematic

Pregnancy combines flow and coagulation changes

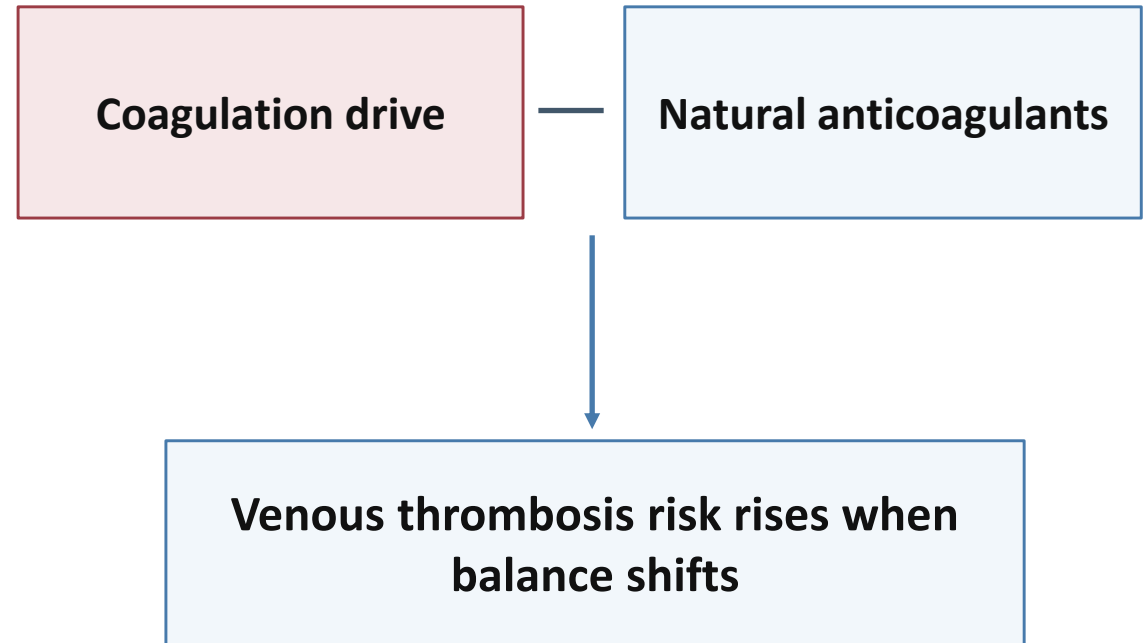
- The gravid uterus impedes venous return from the legs.
- Late pregnancy and the postpartum period also involve systemic hypercoagulability.
- These effects act together, rather than as isolated risk factors.



Multi-hit model; Relationships are qualitative

Estrogenic states favor coagulation

- Oral contraceptive use is an acquired venous-thrombosis risk factor.
- Higher hepatic synthesis of clotting factors and reduced anticoagulant synthesis are implicated.
- The finding illustrates a systemic mechanism without a focal venous lesion.



Qualitative schematic only; Not a measured scale

Obesity and advanced age modify DVT risk

- Obesity is included among acquired clinical risk factors.
- Older age increases DVT risk irrespective of the immediate trigger.
- The source does not require a single specific mechanism for each association.

Inherited thrombophilia changes baseline susceptibility

- Primary hypercoagulability follows inherited coagulation-pathway abnormalities.
- Common examples include factor V Leiden and prothrombin G20210A.
- Rare causes include deficiencies of natural anticoagulants.

Factor V Leiden

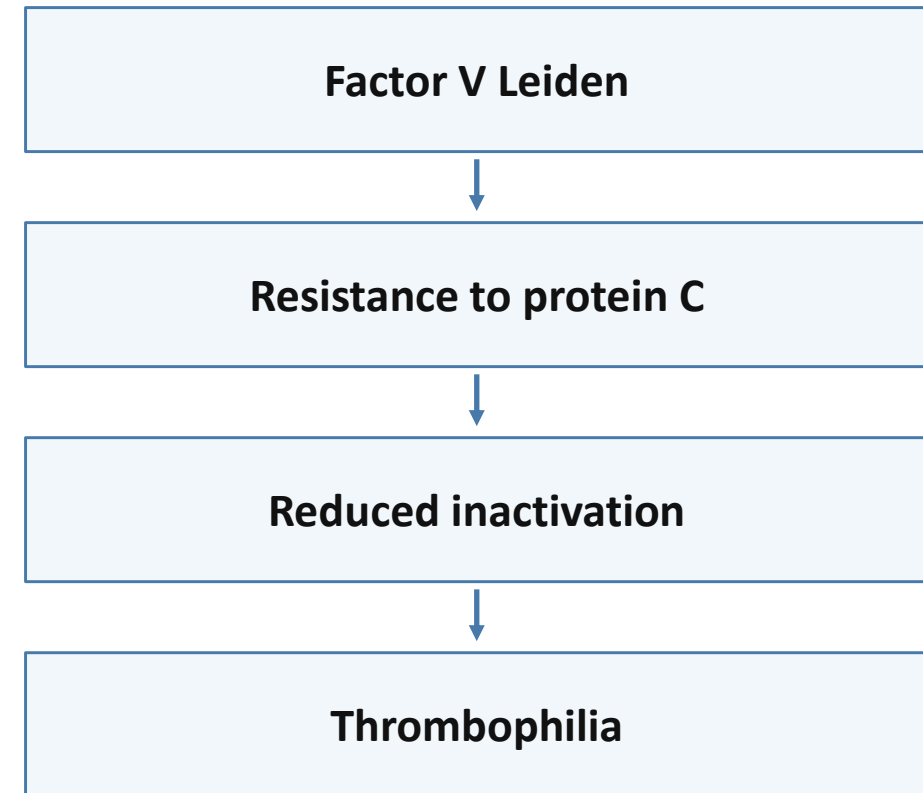
Prothrombin G20210A

Antithrombin deficiency

Protein C / S deficiency

Factor V Leiden impairs normal anticoagulant control

- Mutated factor V resists cleavage and inactivation by activated protein C.
- Activated factor V activity therefore persists longer.
- The net effect is an increased propensity to venous thrombosis.



Original editable causal schematic

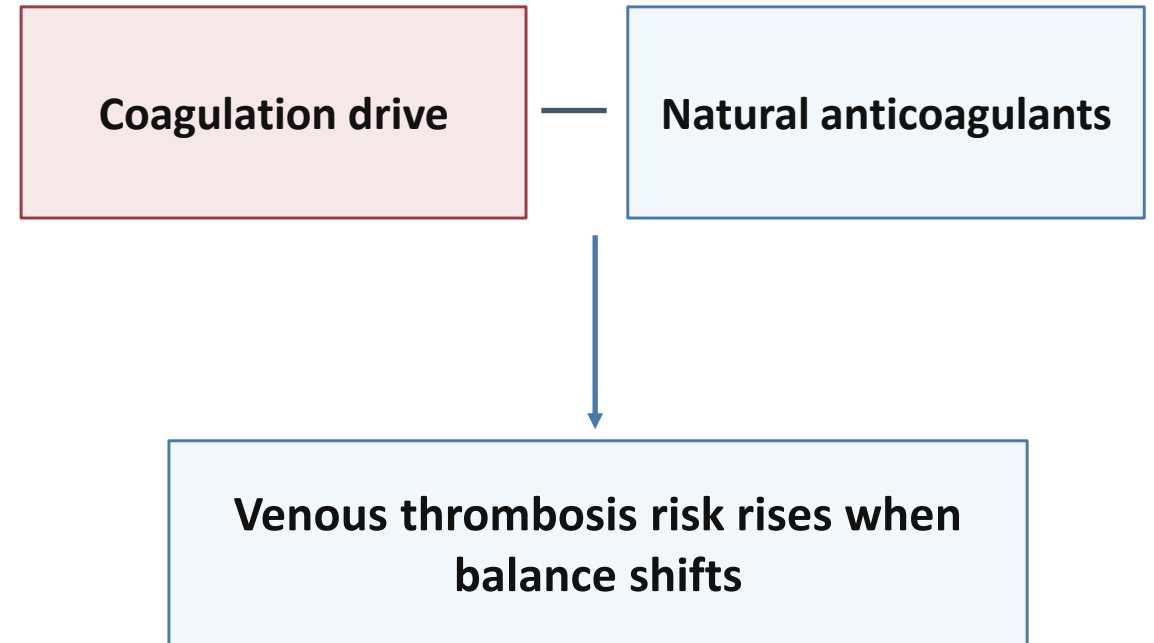
Prothrombin G20210A increases procoagulant substrate

- The 3'-untranslated-region variant is associated with increased prothrombin.
- Greater prothrombin availability favors clot formation.
- It is a distinct inherited mechanism from factor V Leiden.



Deficient endogenous inhibitors permit excess thrombin

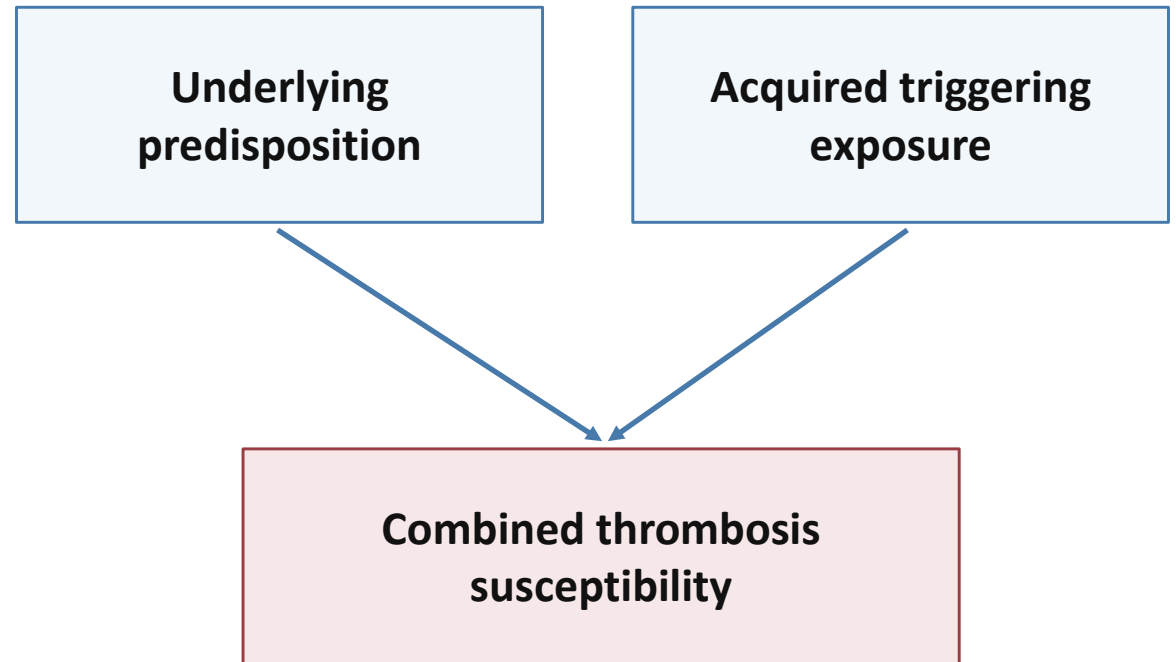
- Antithrombin deficiency reduces physiologic thrombin inhibition.
- Protein C and protein S deficiencies weaken natural anticoagulant activity.
- Affected patients can develop recurrent venous thromboembolism at younger ages.



Qualitative schematic only; Not a measured scale

Inherited and acquired risks can interact

- A thrombophilic genotype does not guarantee a thrombotic event.
- Prolonged immobilization can supply an additional provoking factor.
- The combined-risk model explains events arising after a recognizable trigger.



Multi-hit model; Relationships are qualitative

Systemic hypercoagulability is not only genetic

- Malignancy, pregnancy, estrogen use, and acquired immune-mediated disorders can shift coagulation.
- Local venous stasis may amplify systemic susceptibility.
- Clinically, one event can reflect several causal contributors.

Malignancy

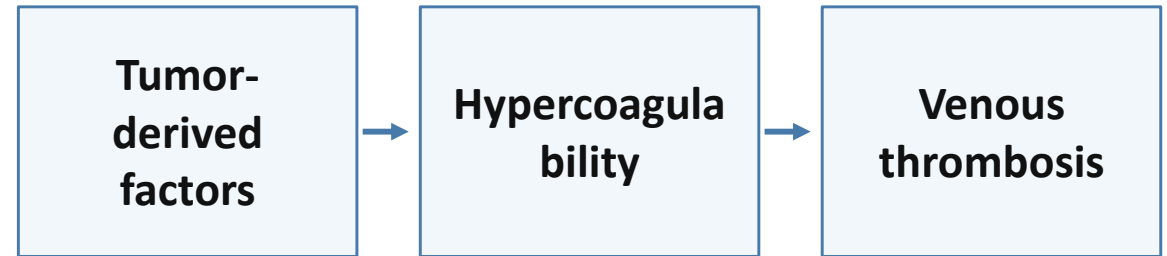
Pregnancy / estrogen

**Antiphospholipid
syndrome**

Immobilization + stasis

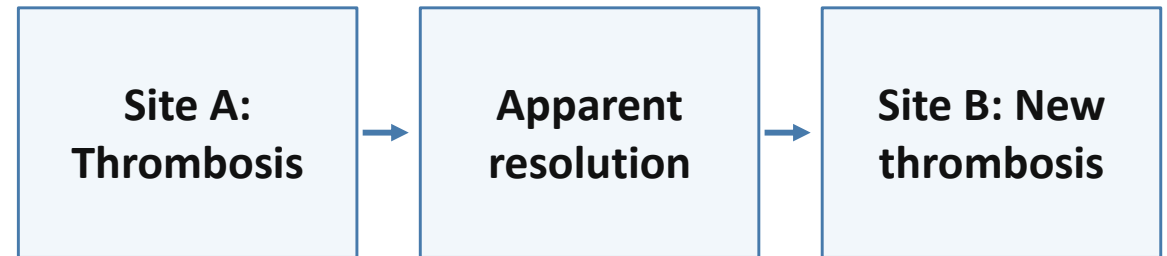
Malignancy produces a prothrombotic state

- Cancer, particularly adenocarcinoma, may induce paraneoplastic hypercoagulability.
- Tumor cells can release procoagulants into the circulation.
- The result may be venous thrombi without an obvious local vessel lesion.



Trousseau syndrome is migratory thrombophlebitis

- A venous thrombus appears at one site, resolves, and a new thrombus develops elsewhere.
- This classic migratory pattern may accompany an underlying carcinoma.
- The term refers to recurrent, anatomically changing thrombotic events.



Migration means recurrence, not one moving clot

- In Trousseau syndrome, successive local venous thromboses arise at different sites.
- By contrast, embolization means actual detachment and transport of a thrombus fragment.
- These are mechanistically distinct clinical descriptions.

Feature	Migratory thrombophlebitis	Thromboembolism
Process	New thromboses at separate sites	Detached clot fragment travels
Underlying issue	Persistent systemic procoagulant drive	Mechanical clot dislodgement
Typical concern	Occult malignancy association	Remote vascular obstruction

Editable comparison table

Antiphospholipid syndrome is another acquired cause

- Antiphospholipid antibodies are associated with recurrent thrombosis.
- Endothelial, platelet, complement, and fibrinolytic pathways may be involved.
- Venous thrombosis can coexist with other systemic manifestations.

Deep veins outside the leg can also thrombose

- Periprostatic veins and female pelvic venous plexuses are recognized sites.
- Upper-extremity veins can also contain venous thrombi.
- Anatomic site alters the local consequences and potential route of embolization.

Periprostatic plexus

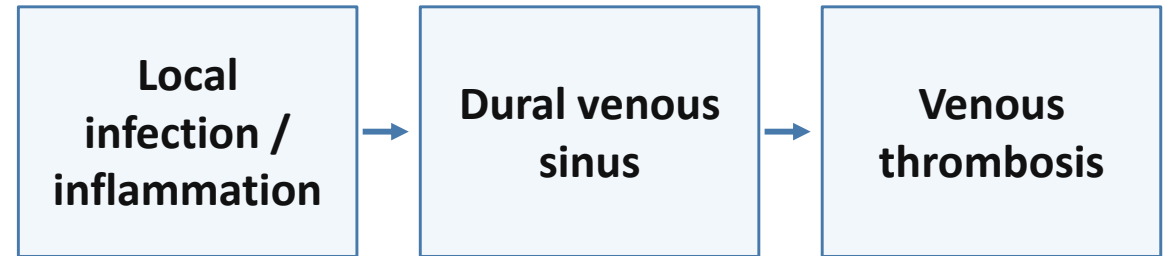
**Pelvic / periuterine
veins**

Upper-extremity veins

**Lower-extremity deep
veins**

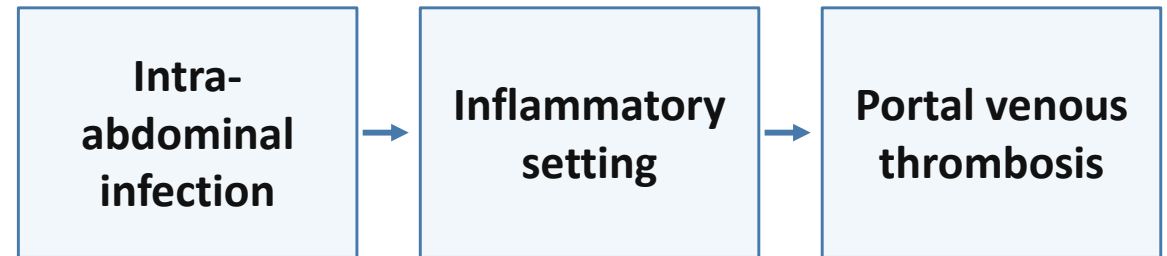
Dural venous sinus thrombosis has a distinct location

- Large veins within the skull and dural sinuses can thrombose.
- Infection or inflammation is a recognized predisposing setting.
- This site differs anatomically from the common lower-extremity DVT.



Portal vein thrombosis can accompany abdominal infection

- Peritonitis, appendicitis, salpingitis, or pelvic abscess may precede portal thrombosis.
- This illustrates venous thrombosis occurring in an inflammatory setting.
- Portal thrombosis can also arise with systemic thrombophilic disorders.

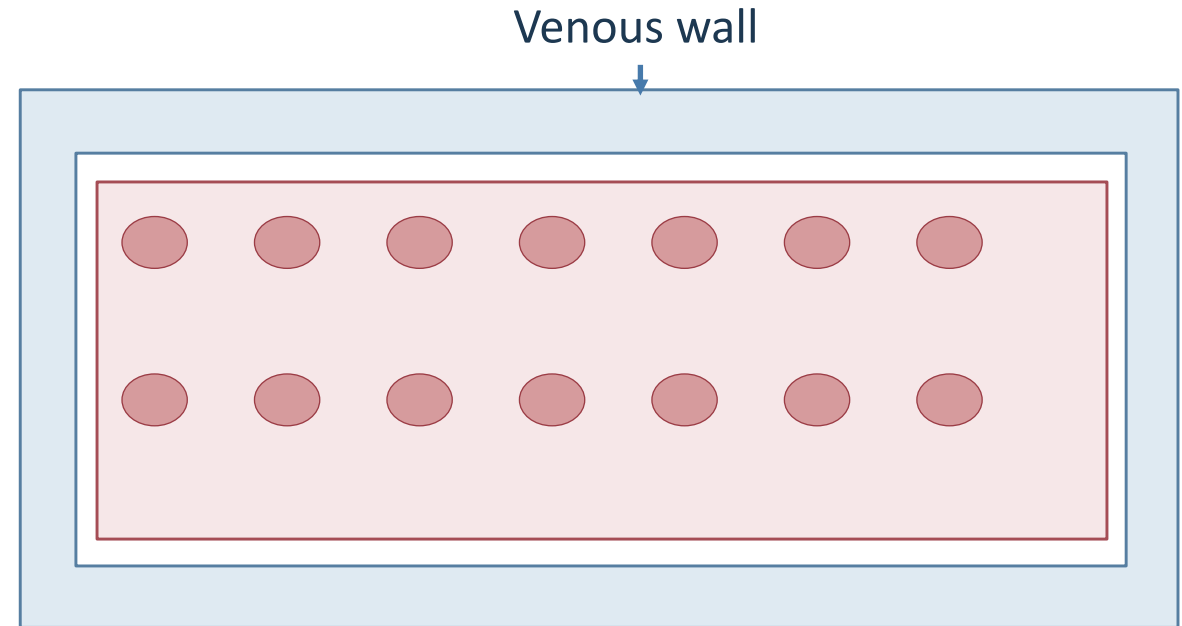


Polycythemia vera adds an unusual venous risk

- Portal thrombosis with thrombophilic states including polycythemia vera.
- Abnormal hematologic states can alter blood rheology or platelet function.
- Consider a systemic driver when thrombosis occurs at an atypical site.

The venous thrombus often forms an occlusive cast

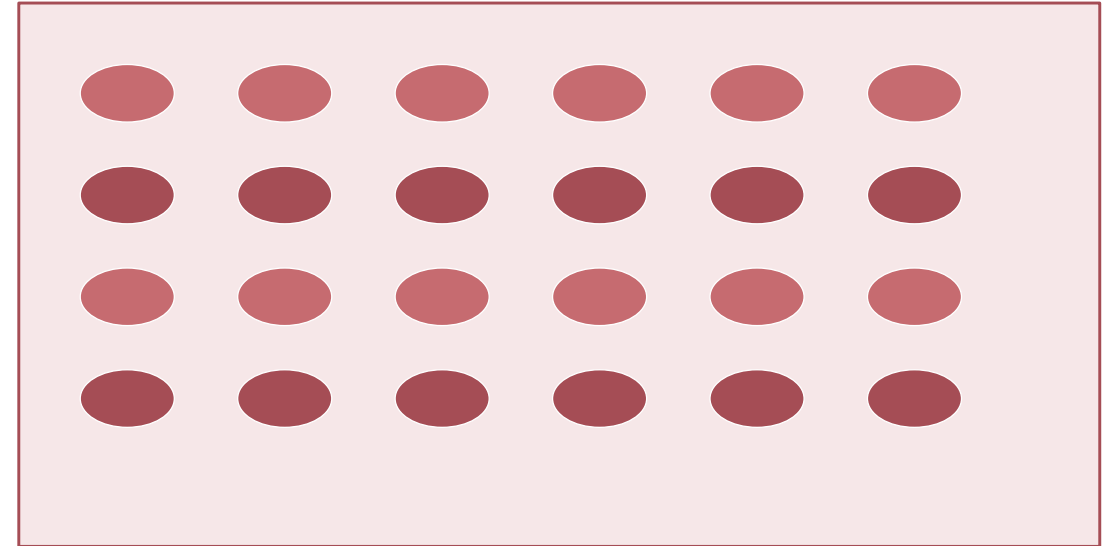
- Venous thrombi are usually nearly or completely occlusive.
- The thrombus may fill a long segment of the venous lumen.
- Grossly it is firm and focally adherent to the vessel wall.



Red cell-rich occlusive cast

Venous thrombi are red-cell rich

- Low-flow venous clots trap many erythrocytes within a fibrin network.
- They contain relatively fewer platelets than arterial thrombi.
- Thus the terms red thrombus and stasis thrombus are used.

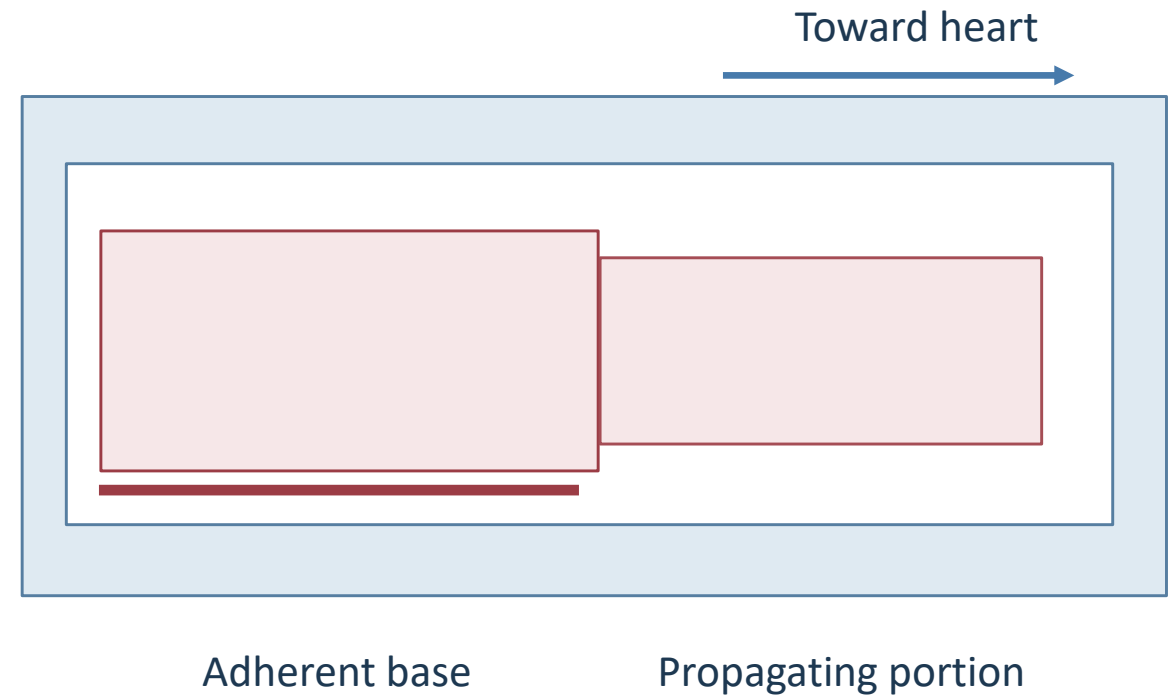


Erythrocytes enmeshed in fibrin

Schematic composition; Relative proportions not quantitative

Site of attachment records thrombus formation in life

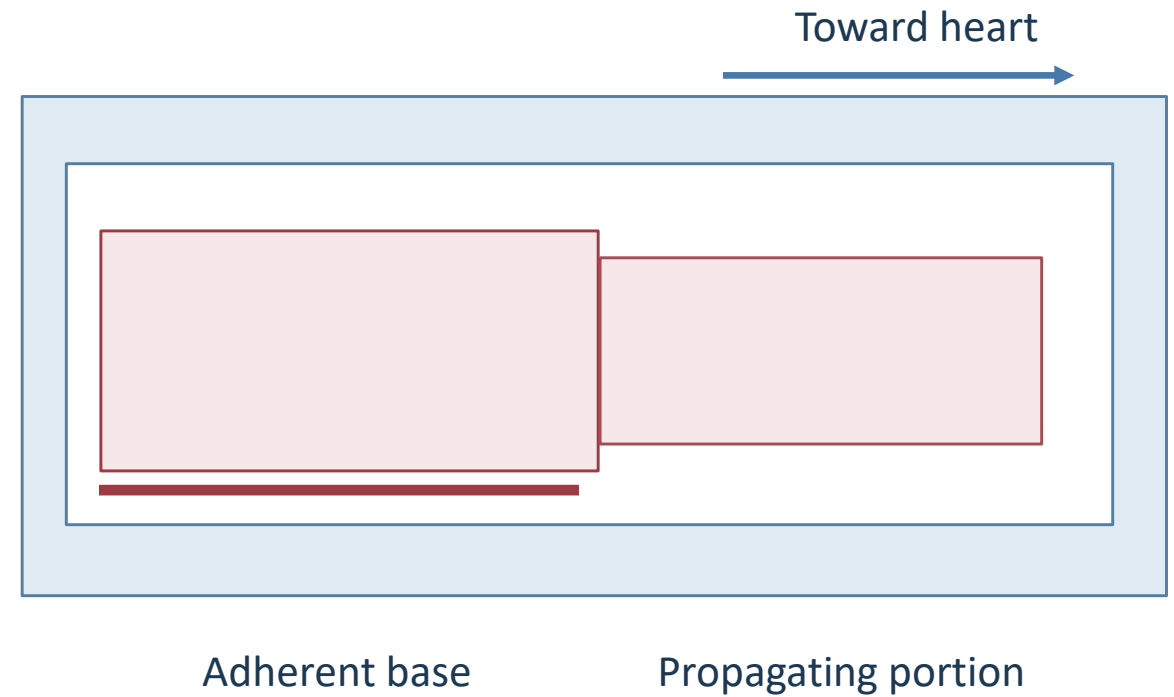
- Antemortem thrombi are usually focally attached to the venous wall.
- The wall attachment marks the site of initiation.
- An unattached tail may present greater embolic potential.



Schematic: Direction and attachment; Not an observed specimen

Venous thrombi may propagate toward the heart

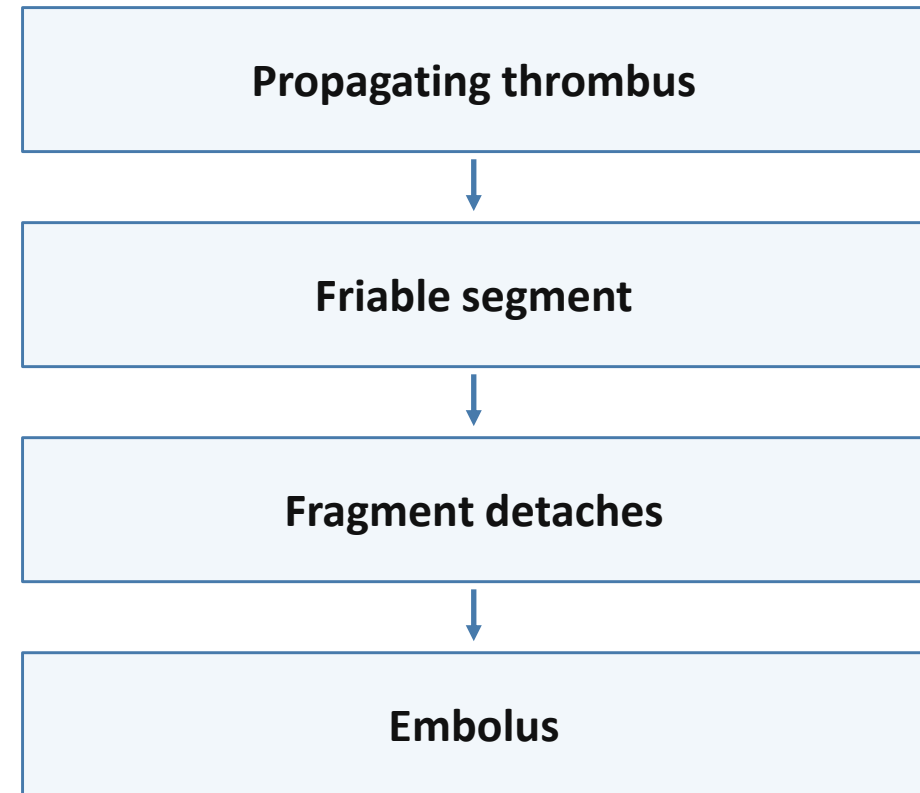
- Venous thrombi tend to extend in the direction of blood flow.
- This growth can produce a longer luminal thrombus.
- The propagating portion may remain relatively weakly attached.



Schematic: Direction and attachment; Not an observed specimen

Poorly attached portions may fragment

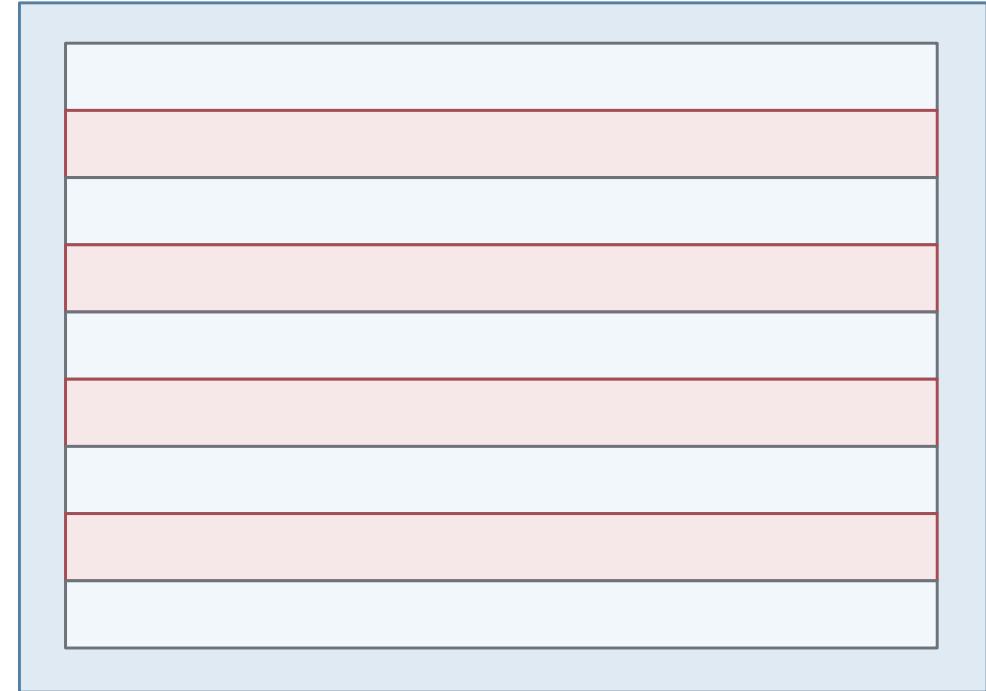
- Propagation increases the mass of the venous thrombus.
- A weakly adherent distal portion can detach.
- Once detached, it becomes a thromboembolus and follows venous flow.



Original editable causal schematic

Lines of Zahn show laminated thrombus formation

- Alternating pale platelet–fibrin layers and darker erythrocyte-rich layers may be seen.
- These laminations indicate formation in flowing blood.
- They can help distinguish antemortem thrombi from postmortem clots.



Pale platelet / fibrin

Dark red-cell layer

Schematic laminations — not a microscopy image

Antemortem thrombus versus postmortem clot

- Antemortem: Firm, attached to wall, and may show lamination.
- Postmortem: Gelatinous, typically not wall-attached.
- Postmortem sedimentation produces dependent dark-red and upper yellow portions.

Criterion	Antemortem venous thrombus	Postmortem clot
Consistency	Firm	Gelatinous
Wall relationship	Focal attachment	Usually unattached
Internal pattern	Possible lines of Zahn	Red dependent / yellow upper layers

Editable comparison table

Arterial and venous thrombi differ in dominant drivers

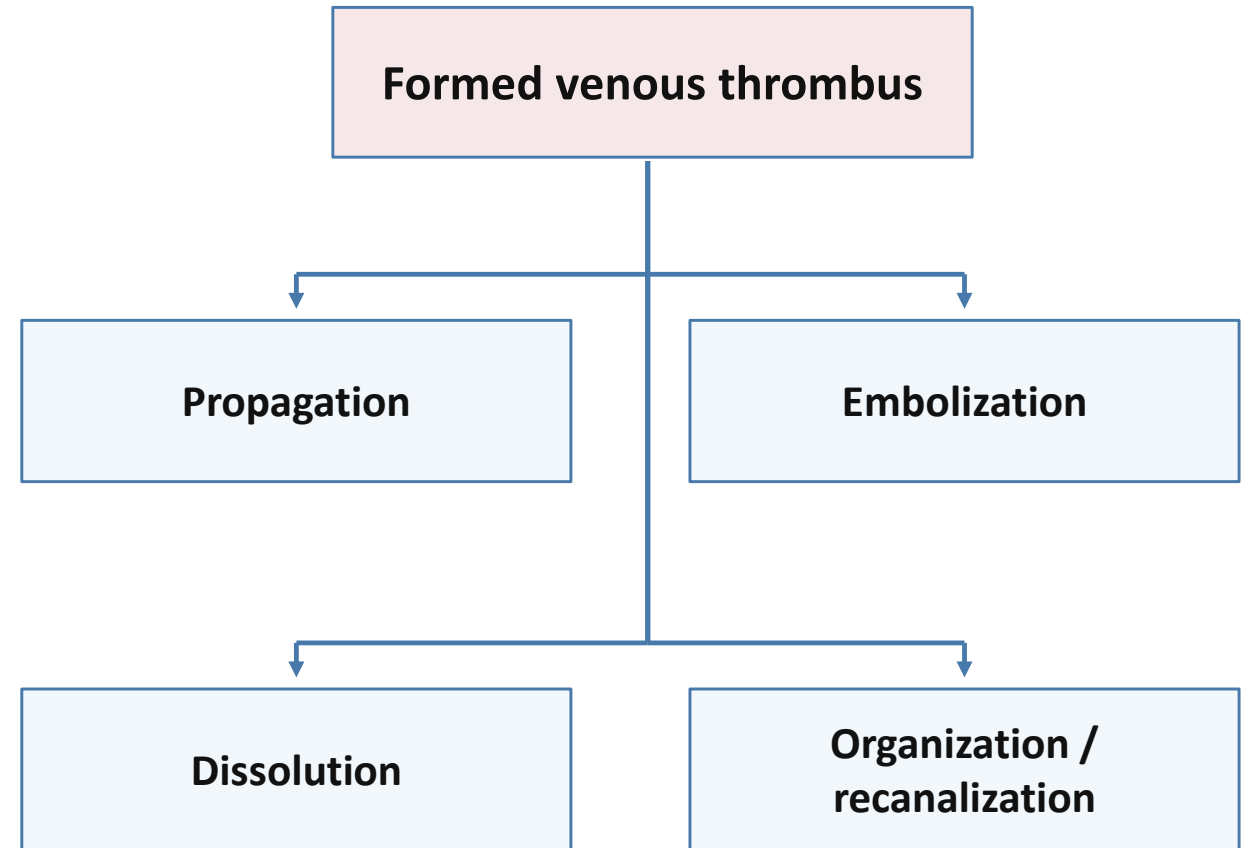
- Arterial thrombi typically begin at endothelial injury or turbulence.
- Venous thrombi characteristically form at sites of stasis.
- Arterial thrombi are generally more platelet-rich; Venous thrombi more erythrocyte-rich.

Dimension	Venous thrombosis	Arterial thrombosis
Predominant flow factor	Stasis	Turbulence / injury
Composition	More red cells	More platelets
Clinical concern	Venous congestion / pulmonary embolus	Downstream ischemia / infarction

Editable comparison table

A thrombus can follow four main paths

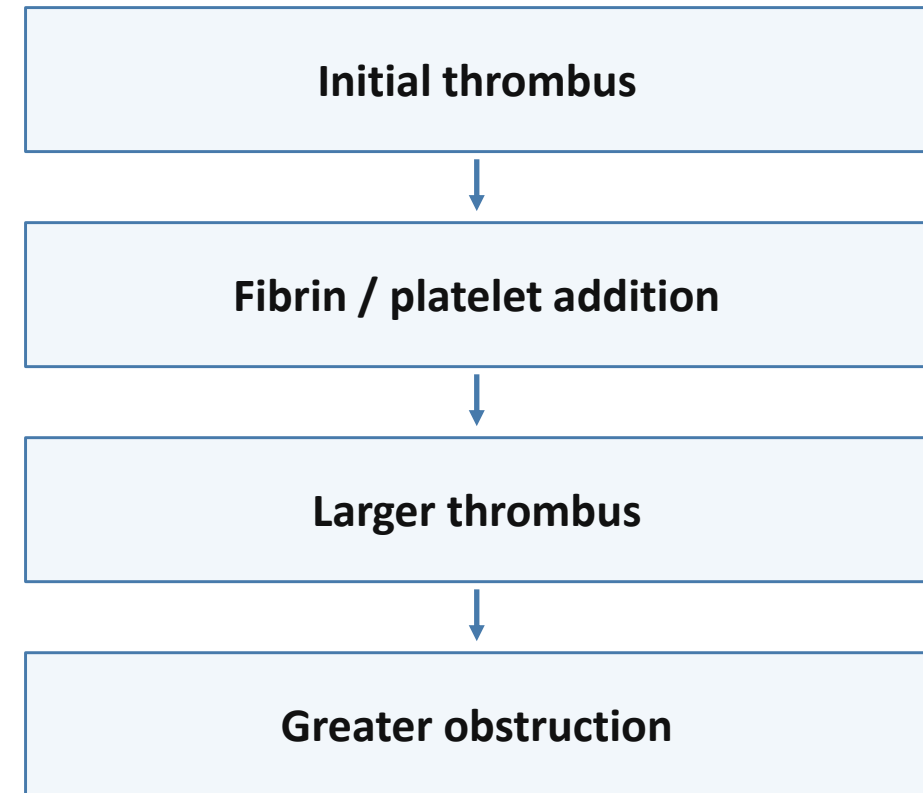
- Propagation: Ongoing addition of fibrin and platelets.
- Embolization: Detachment and movement to another vascular bed.
- Dissolution: Fibrinolysis of thrombotic material.
- Organization and recanalization: Cellular ingrowth and channel formation.



Four nonexclusive fates

Propagation can deepen venous obstruction

- New fibrin and platelets are incorporated into the existing thrombus.
- Thrombus length and degree of luminal blockage may increase.
- Extension may also enlarge the portion vulnerable to embolization.



Original editable causal schematic

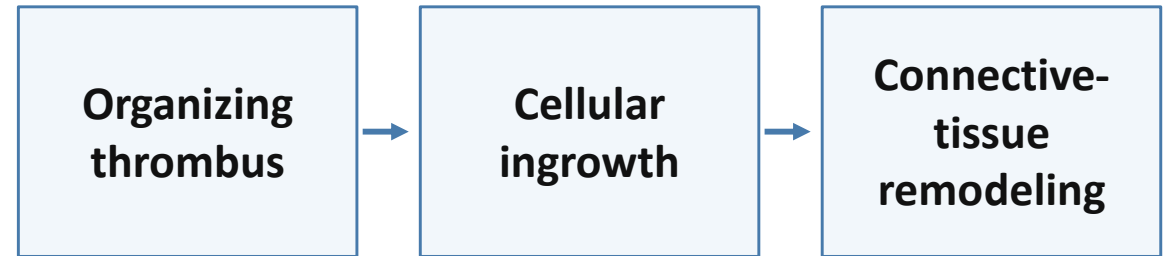
Fibrinolysis can remove a recent thrombus

- Activation of plasmin-mediated fibrinolysis may shrink a recent thrombus.
- Older thrombi contain more cross-linked fibrin and resist lysis.
- This difference explains why thrombus age influences reversibility.



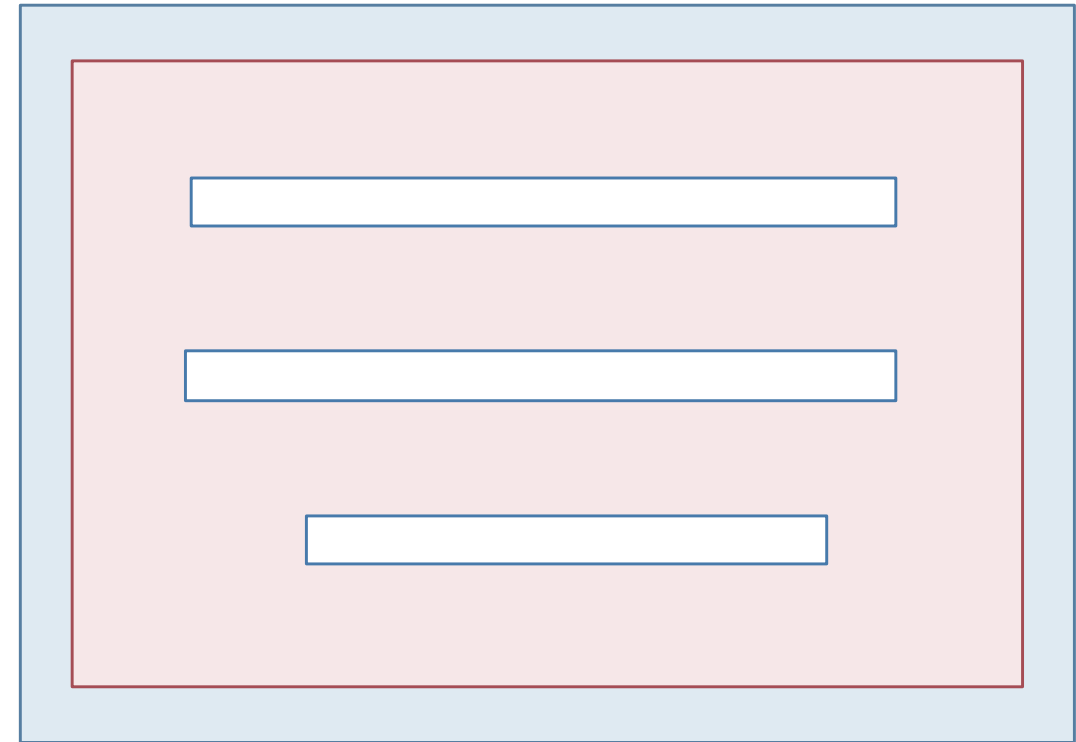
Organization converts thrombus to vascular tissue

- Endothelial cells, smooth-muscle cells, and fibroblasts grow into the clot.
- The original thrombus becomes progressively incorporated into the wall.
- This is a reparative tissue response, not simply blood-clot persistence.



Recanalization can partially restore blood flow

- Small vascular channels form within an organized thrombus.
- These channels may reestablish continuity of the original lumen.
- Restoration is variable, and residual fibrotic narrowing may persist.

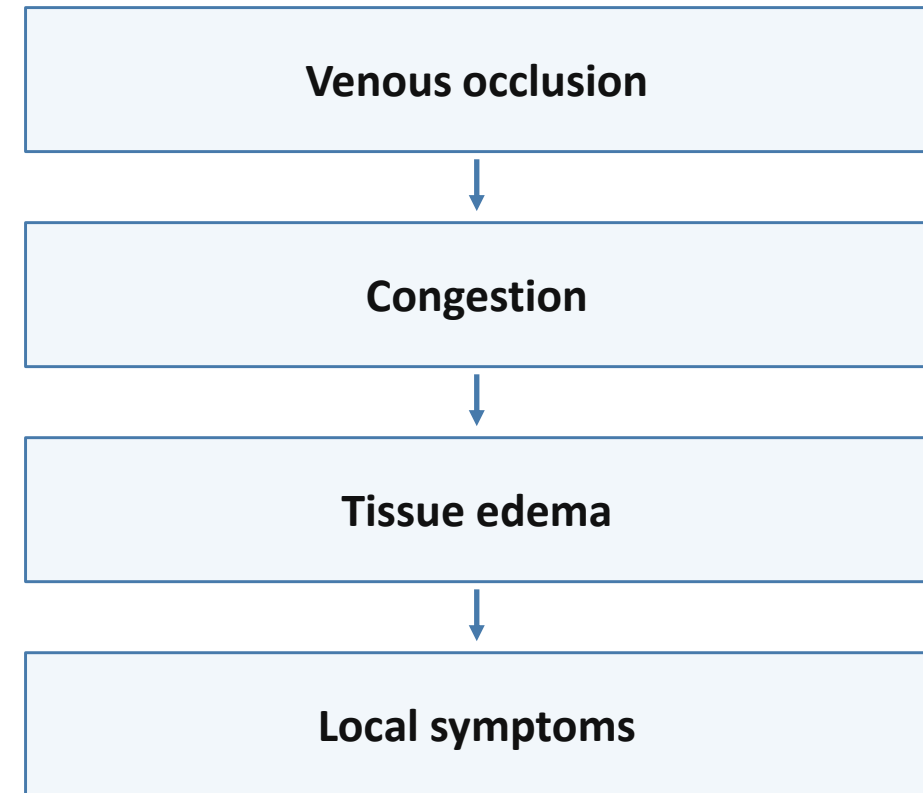


New luminal channels

Schematic recanalization; Restored flow may be incomplete

Venous blockage produces pressure-related effects

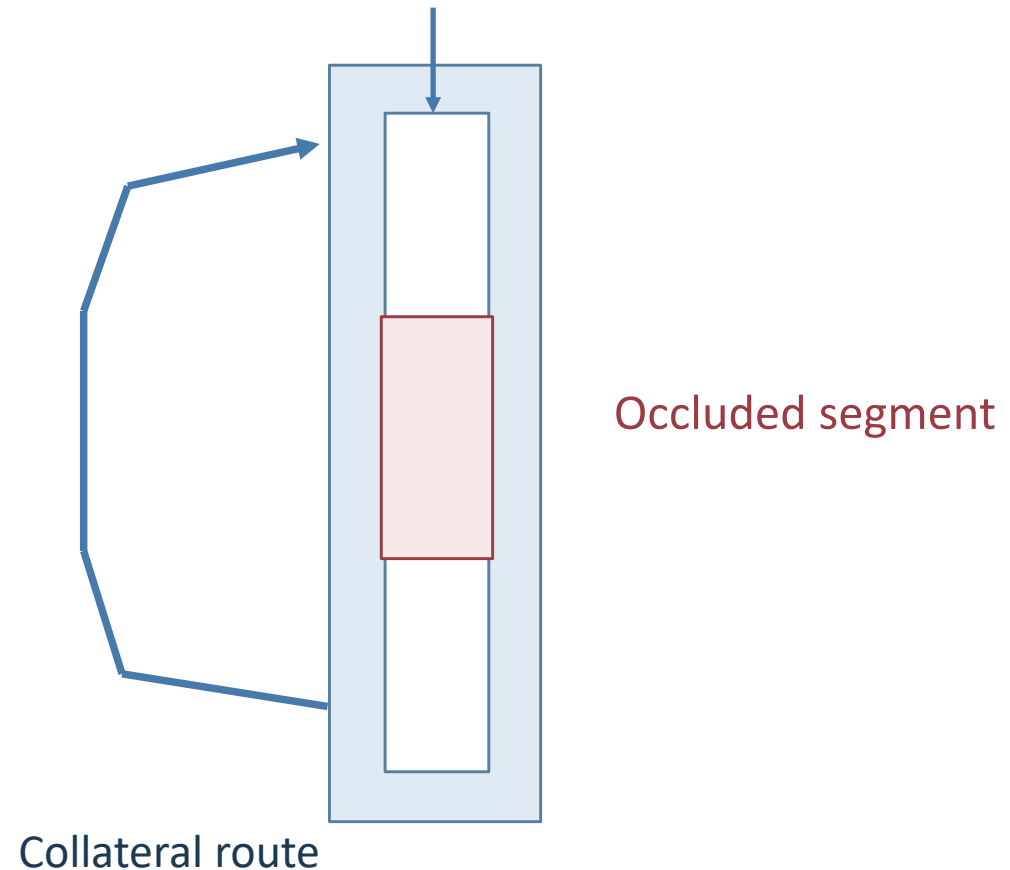
- Outflow obstruction increases venous congestion distal to the clot.
- Higher hydrostatic pressure promotes tissue edema.
- Pain and swelling may occur, depending on the site and collateral drainage.



Original editable causal schematic

Collateral veins can conceal DVT symptoms

- Alternate venous channels may open as the principal vein becomes blocked.
- Collateral drainage reduces overt congestion and edema.
- The absence of dramatic local findings therefore does not imply absence of thrombus.



Original schematic — collateral flow may mask symptoms

DVT can be clinically silent

- Leg thrombi often produce few reliable local signs or symptoms.
- Chapter 4 describes approximately half of DVTs as asymptomatic.
- Pulmonary embolism may be the first recognized manifestation.

Local findings are suggestive, not dependable

- Potential findings include venous dilation, edema, cyanosis, warmth, erythema, and pain.
- Any of these findings may be absent in a patient with DVT.
- Clinical inspection alone cannot establish or exclude a thrombus.

Edema / venous dilation

Pain / tenderness

Warmth / erythema

Cyanosis

Homan sign cannot exclude DVT

- Pain with forced dorsiflexion has historically been called Homan sign.
- Calf compression or pressure over veins can also provoke discomfort.
- Absence of these findings does not exclude DVT; They are unreliable clinical signs.

Superficial venous thrombosis has a different hazard profile

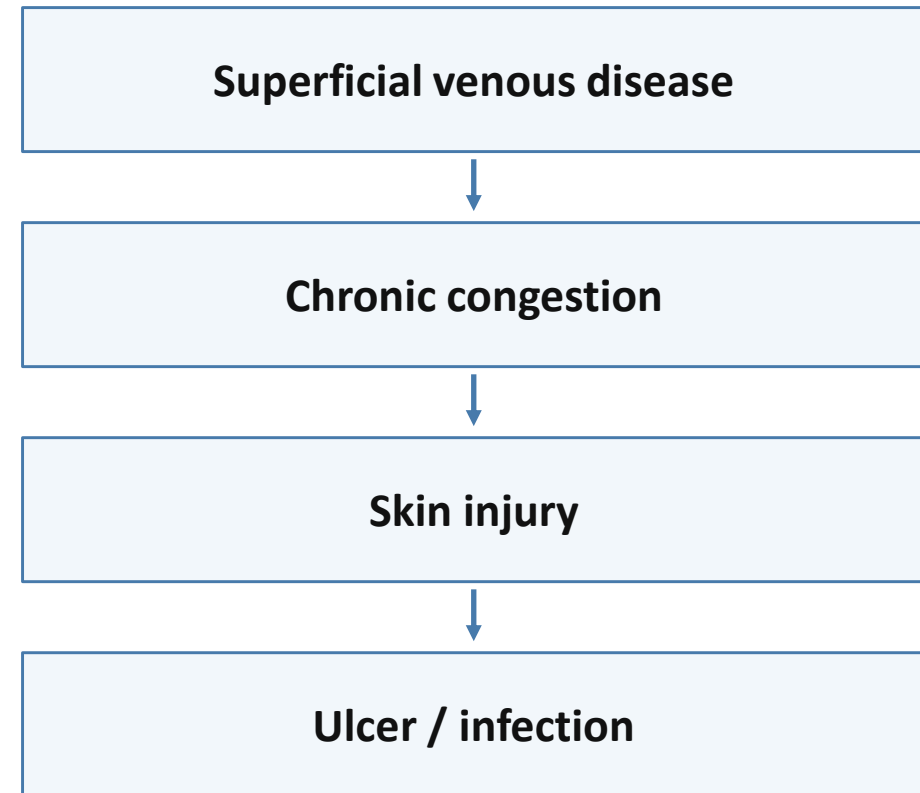
- Thrombosed superficial saphenous veins may be painful and tender.
- Local congestion and edema can impair superficial tissue drainage.
- Embolization from these veins is much less common than from deep veins.

Presentation	Superficial veins	Deep veins
Local symptoms	Often prominent	May be absent
Superficial skin change	Congestion / ulcer risk	Not the defining feature
Pulmonary embolization	Rare	Major complication

Editable comparison table

Chronic superficial venous congestion harms skin

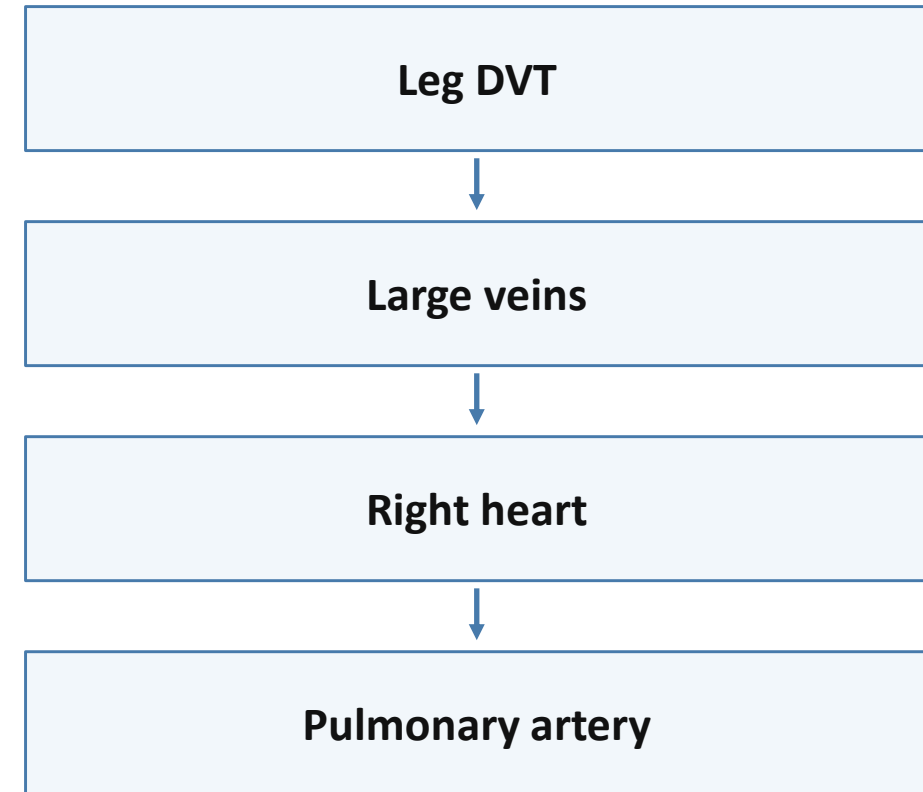
- Impaired venous outflow can cause sustained congestion and edema.
- Stasis dermatitis and skin ulceration may complicate associated varicosities.
- Poor healing and secondary infection compound tissue injury.



Original editable causal schematic

DVT causes pulmonary embolism by clot detachment

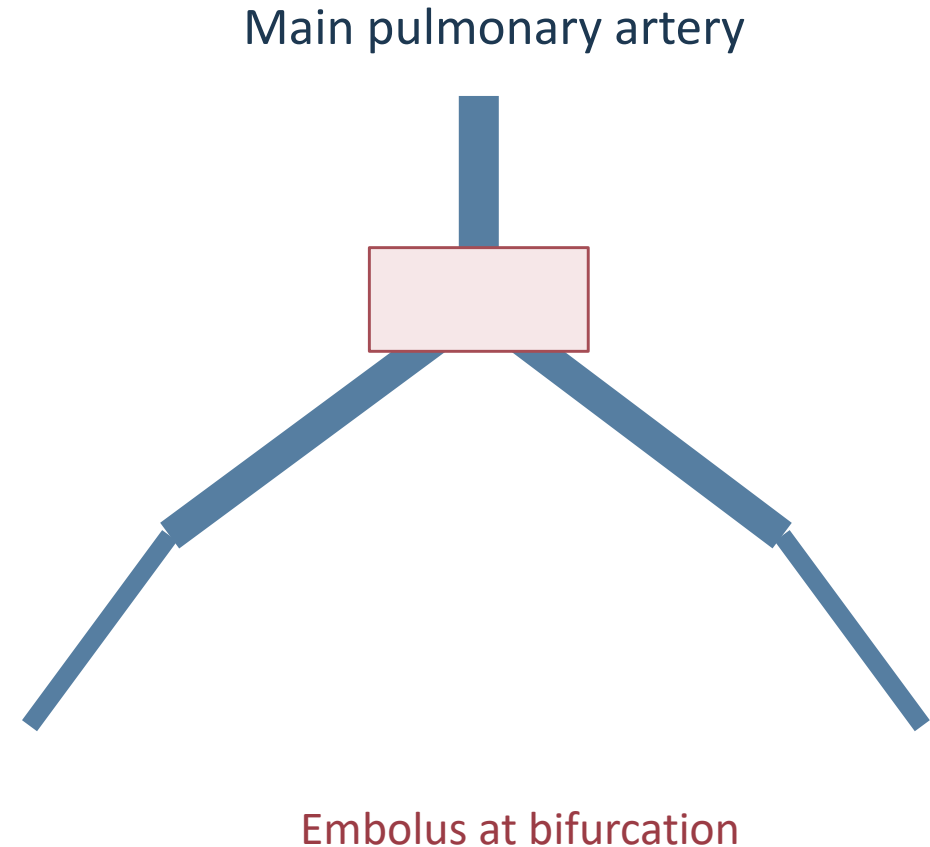
- A fragment of deep venous thrombus separates from its wall attachment.
- It travels through progressively larger veins and the right heart.
- It lodges in a pulmonary arterial vessel too small for onward passage.



Original editable causal schematic

Pulmonary embolus location depends on fragment size

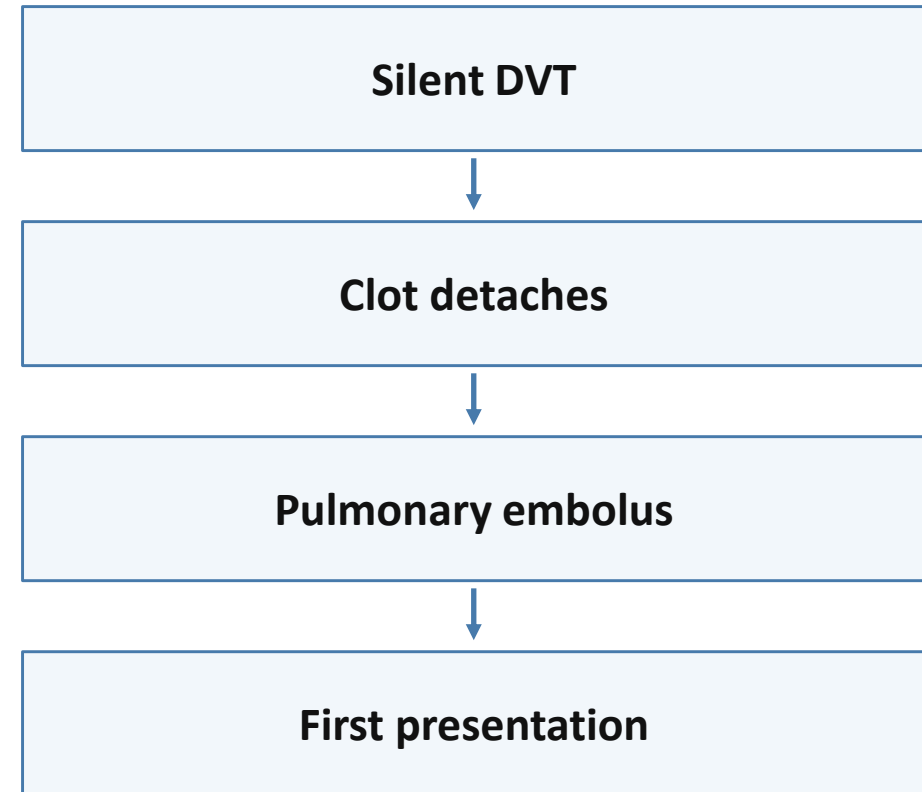
- A large embolus may block the main pulmonary artery or straddle its bifurcation.
- Smaller fragments lodge in branching pulmonary arterial vessels.
- Several fragments can present simultaneously or sequentially.



Schematic location only; Not a radiologic image

Pulmonary embolism may reveal a previously silent DVT

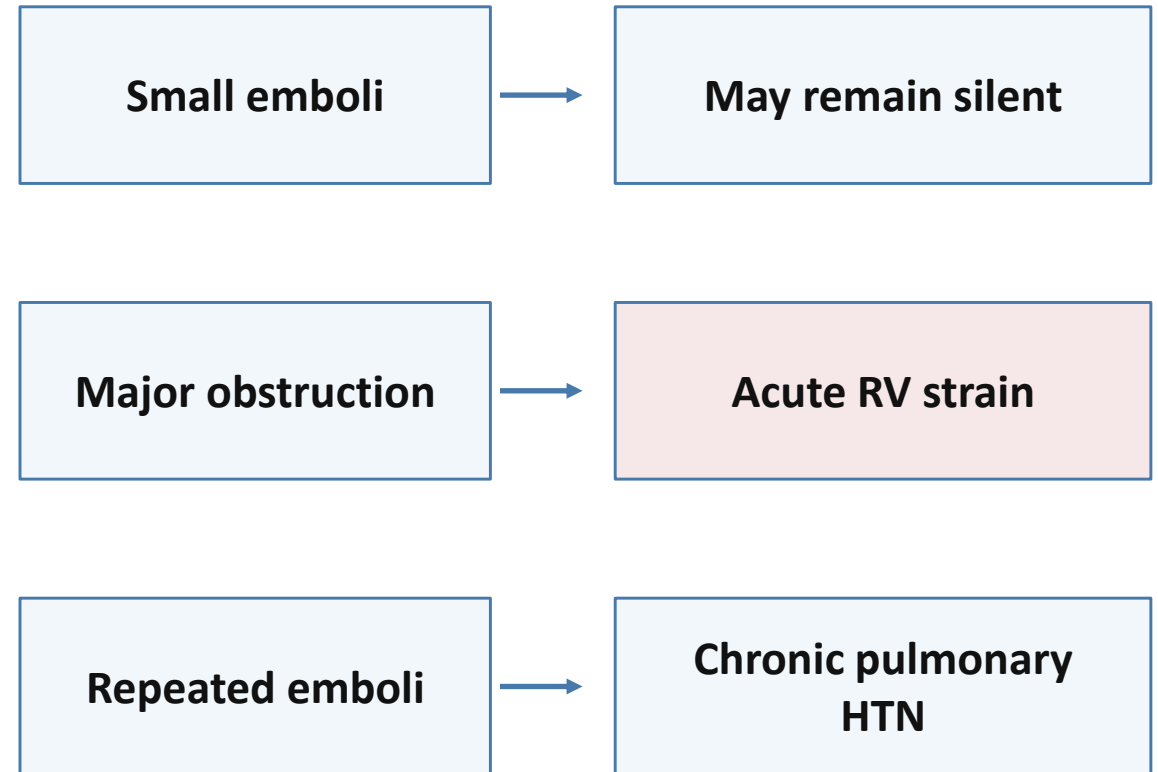
- An embolic event can be the first clinical evidence of thrombophlebitis.
- The affected leg may have lacked recognizable symptoms.
- This disconnect explains the importance of the embolic complication.



Original editable causal schematic

Pulmonary embolic burden determines the outcome

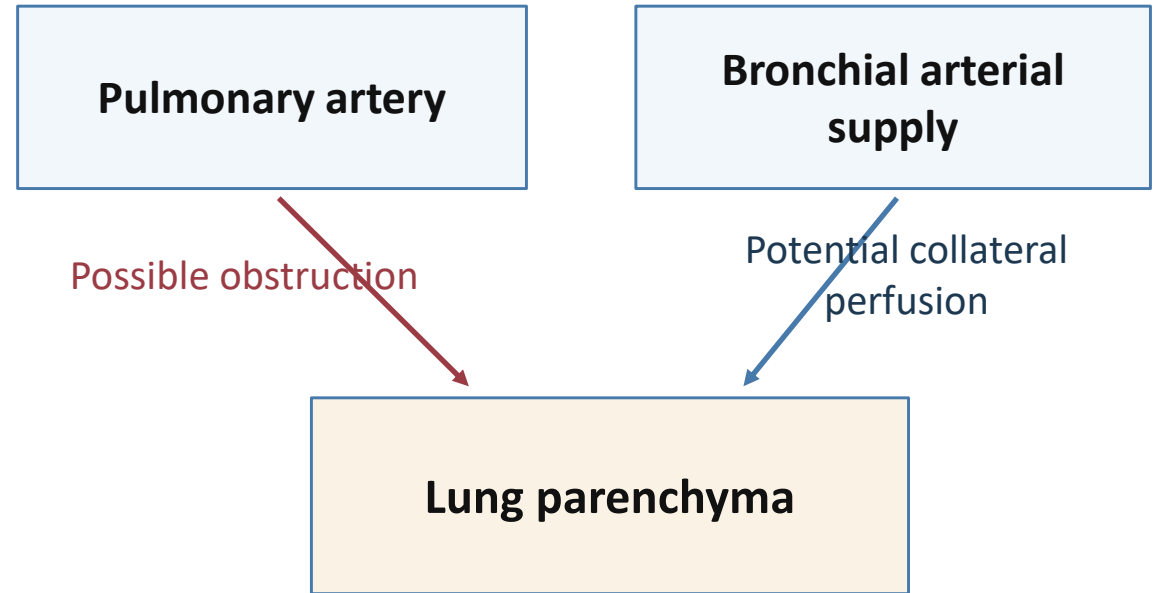
- Smaller emboli may produce no clinical manifestations.
- Major pulmonary vascular obstruction can induce acute right-heart failure or cardiovascular collapse.
- The outcome varies with embolus size, number, and the affected vascular bed.



Qualitative consequences; Not a severity scoring system

Pulmonary hemorrhage and infarction are not equivalent

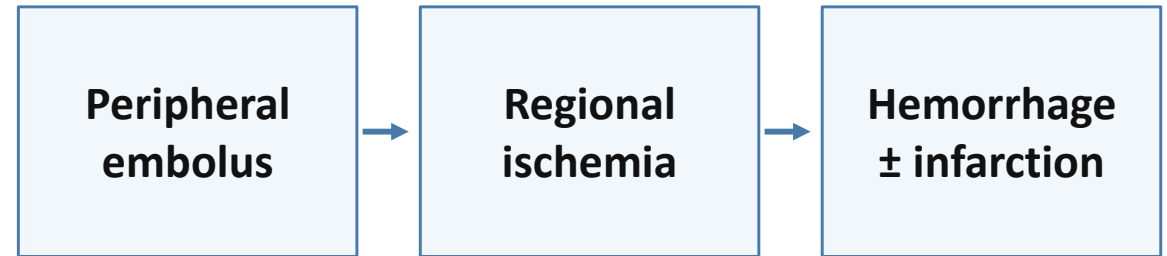
- The lung has both pulmonary and bronchial arterial supplies.
- Bronchial flow may preserve viability despite pulmonary arterial blockage.
- Infarction becomes more likely when bronchial perfusion is compromised.



Conceptual dual supply — infarction is context-dependent

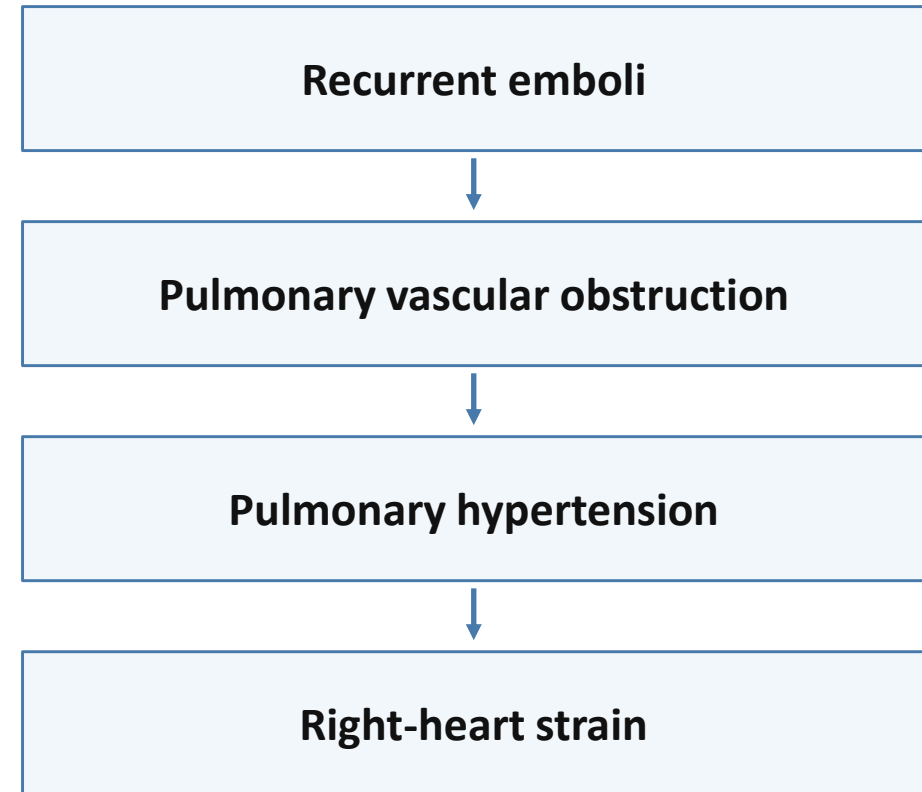
Small-vessel emboli may cause pulmonary infarction

- Occlusion of smaller end-arteriolar branches may produce hemorrhage or infarction.
- Pulmonary infarcts are commonly hemorrhagic because of dual blood supply and tissue structure.
- The vascular site and remaining perfusion determine tissue outcome.



Recurrent emboli can produce pulmonary hypertension

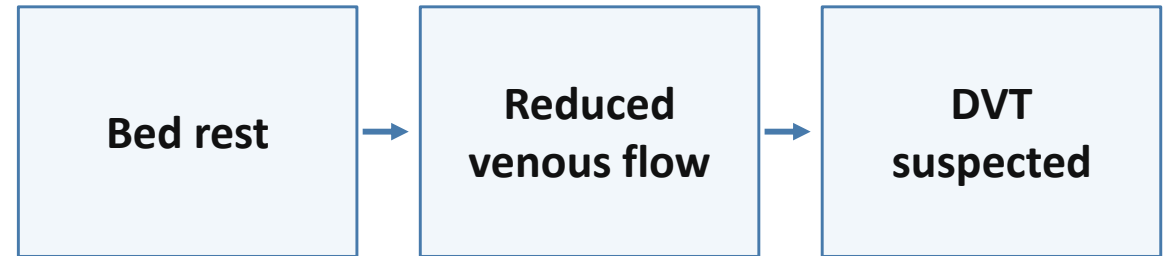
- Repeated pulmonary embolization can chronically obstruct pulmonary vessels.
- Elevated resistance to pulmonary flow develops.
- Pulmonary hypertension may ultimately strain or fail the right ventricle.



Original editable causal schematic

Case: Immobilization and unilateral leg symptoms

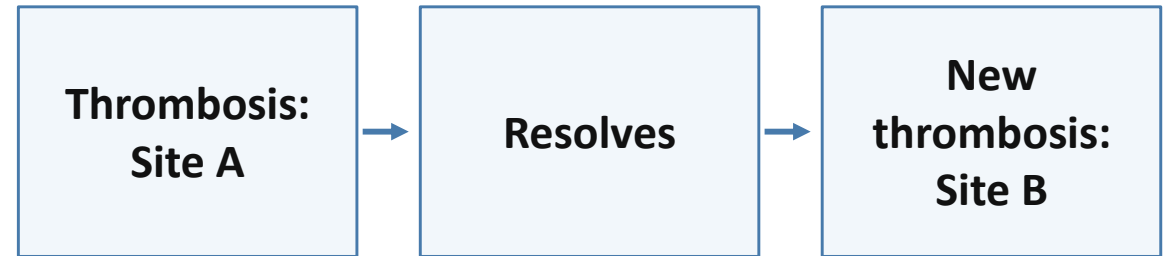
- A hospitalized patient has prolonged bed rest and new asymmetric leg swelling.
- Identify the dominant element of Virchow triad: Venous stasis.
- Explain why the absence of calf pain would not exclude the suspected thrombus.



Which Virchow factor dominates?

Case: Recurrent thrombosis at changing sites

- A patient develops superficial venous thromboses at anatomically separate sites over time.
- The pattern suggests migratory thrombophlebitis.
- Link the clinical pattern to a persistent systemic procoagulant state, classically malignancy.



Why do anatomically separate events recur?

Case: Sudden pulmonary event without leg findings

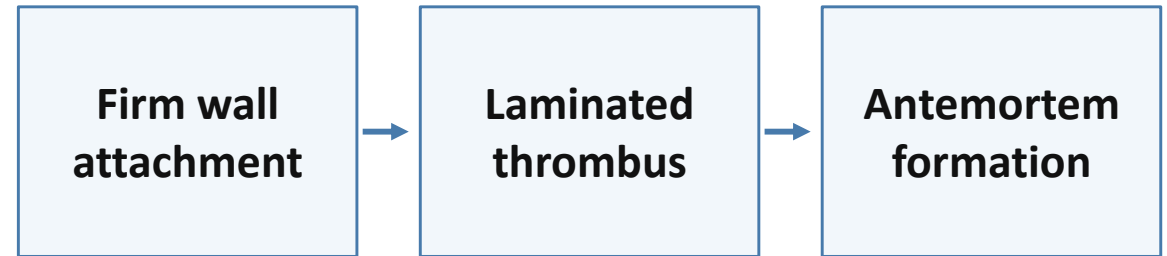
- A patient develops sudden cardiopulmonary compromise but had no obvious leg signs.
- A deep venous thrombus may have been clinically silent.
- A detached fragment may have obstructed the pulmonary arterial circulation.



Why could leg signs have been absent?

Case: Firm venous cast at autopsy

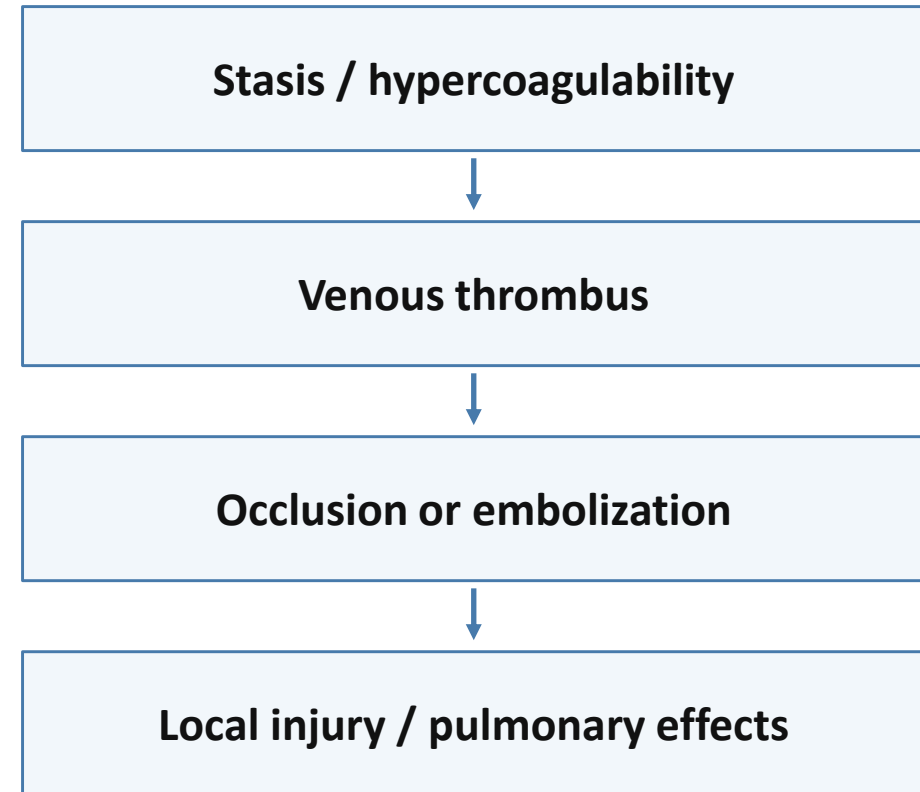
- A firm red luminal cast is focally adherent to the venous wall.
- Alternating pale and dark layers are visible in the clot.
- This combination supports an antemortem venous thrombus rather than a postmortem clot.



Which findings support antemortem formation?

Integrated clinicopathologic model

- Predisposition: Stasis and/or hypercoagulability, sometimes with endothelial dysfunction.
- Lesion: An adherent, erythrocyte-rich, often occlusive venous thrombus.
- Consequences: Local congestion, silent DVT, embolization, or organization.
- Critical danger: Pulmonary embolism and possible chronic pulmonary hypertension.



Original editable causal schematic

References and source limits

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. 2026. Chapter 11: Blood Vessels, pp. 471–472.
- Same textbook, Chapter 4: Hemodynamic Disorders, Thromboembolic Disease, and Shock, pp. 109–115.
- Teaching material drawn from the supplied topic Markdown and robbins.md; original chapter PDF was not supplied.
- Schematic diagrams are editable conceptual illustrations; no patient specimens are represented.